International Journal of Science and Research (IJSR)
ISSN: 2319-7064
Impact Factor 2025: 7.089

# Prevalence of Bone Mineral Disorders in Hemodialysis Patients: A Prospective Observational Study from a Tertiary Care Hospital in South India

*Running Head:* BMD Disorders in Hemodialysis Patients

**Dr. Nandu Ernest[1], Dr. Sridhara K M[2]**

[1]DNB (General Medicine), Department of General Medicine, Chinmaya Mission Hospital, Indiranagar, Bengaluru – 560038, Karnataka, India

[2]Thesis Guide, Department of General Medicine, Chinmaya Mission Hospital, Indiranagar, Bengaluru – 560038, Karnataka, India

**Abstract:** ***Background: Chronic kidney disease–mineral and bone disorder (CKD-MBD) is nearly universal among patients undergoing maintenance hemodialysis and contributes substantially to fracture risk and cardiovascular morbidity. Data on its prevalence and biochemical correlates from Indian dialysis populations remain limited. Methods: This prospective observational study enrolled 85 adult patients undergoing maintenance hemodialysis (>6 months) at Chinmaya Mission Hospital, Bengaluru. Bone mineral density (BMD) was assessed using Lumbar spine DEXA, and bone turnover was categorised using intact parathyroid hormone (iPTH). Serum calcium, phosphate, alkaline phosphatase, and vitamin D were measured. Associations were analysed using Chi-square/Fisher's exact tests (STATA v16.0); p<0.05 was considered significant. Results: Mean age skewed older, with 51.8% of participants aged ≥60 years, and 64.7% were male. Low BMD (osteopenia or osteoporosis) was present in 48.2% of patients (osteopenia 34.1%, osteoporosis 14.1%). Bone turnover by iPTH was heterogeneous (low turnover 33.0%, normal 38.8%, high 28.2%). Vitamin D deficiency (<10 ng/mL) and insufficiency (10–29 ng/mL) were highly prevalent (28.2% and 51.8% respectively). Serum phosphate showed a statistically significant association with BMD status (p=0.04), with hyperphosphatemia more frequent in patients with normal BMD. Vitamin D, serum calcium, alkaline phosphatase, dialysis duration, and comorbidities showed no significant association with BMD or bone turnover status (all p>0.05). Conclusion: Nearly half of hemodialysis patients in this cohort had reduced bone mineral density, with disordered phosphate metabolism emerging as the dominant correlate. Routine biochemical and BMD surveillance, with a particular focus on phosphate control, is warranted in this high-risk population.***

**Keywords:** Chronic kidney disease-mineral bone disorder; hemodialysis; bone mineral density; parathyroid hormone; vitamin D; phosphate

## 1. Introduction

Chronic kidney disease (CKD) affects nearly 1 in 10 people worldwide and carries substantial health, social, and economic burden.[1] Chronic kidney disease–mineral and bone disorder (CKD-MBD) is one among the most complex syndromes accompanying progressive renal dysfunction, and per KDIGO 2017 guidelines it is defined as a systemic disorder of calcium, phosphate, parathyroid hormone (PTH), and vitamin D metabolism, together with abnormalities of bone turnover, mineralisation, and extraskeletal calcification.[2]

CKD-MBD is nearly universal in patients on maintenance hemodialysis. Phosphate retention, driven by declining glomerular filtration, is an early and central abnormality that stimulates FGF-23 and secondary hyperparathyroidism, while intermittent dialysis itself perturbs the calcium–phosphate–PTH axis.[3] Despite advances in dialysis technology, the burden of CKD-MBD remains high and is a key driver of fractures, vascular calcification, and cardiovascular events.

Reported prevalence estimates for bone mineral disorders in hemodialysis populations vary widely (40–88%) depending on region and diagnostic method, and secondary hyperparathyroidism is reported in 40–60% of long-term dialysis patients.[4] Indian data on this subject remain sparse. We therefore undertook this study to assess the prevalence of bone mineral disorders and their biochemical correlates among hemodialysis patients at our centre.

### *Aim*

To determine the prevalence of bone mineral disorders (BMD) in patients undergoing hemodialysis at Chinmaya Mission Hospital, Bengaluru, and to study their association with biochemical markers of mineral metabolism.

## 2. Materials and Methods

### *Study design and setting*

Prospective observational study conducted in the Department of General Medicine, Chinmaya Mission Hospital, Bengaluru, over 12 months.

### *Participants*

- Inclusion: Adults ≥18 years on hemodialysis for >6 months.
- Exclusion: Age <18 years; dialysis <6 months; acute kidney injury; parathyroid/cutaneous/mammary/renal malignancy; hypo-/hyperthyroidism; consent not given.

### *Sample size*

Based on a reported BMD-disorder prevalence of 87.5%,[5] 95% confidence level and 10% absolute precision, the minimum required sample size was 85.

***Study procedure***

- Written informed consent obtained per institutional ethics committee guidelines.
- Structured proforma for demographics and dialysis-related history.
- Bone mineral density: lumbar spine DEXA scan.
- Biochemistry: serum calcium, phosphate, alkaline phosphatase (ALP), intact PTH (iPTH), and 25-hydroxyvitamin D.
- Bone turnover was categorised by iPTH as low (<150 pg/mL), normal (150–300 pg/mL), or high (>300 pg/mL); BMD was categorised by T-score as normal (≥−1.0) or low (osteopenia/osteoporosis, <−1.0).

***Statistical analysis***

Analysis was performed using STATA v16.0. Categorical variables were summarised as frequencies/percentages. Associations between categorical variables were tested using the Chi-square test (or Fisher's exact test where expected cell counts were <5). A subgroup analysis of biochemical associations with bone turnover was additionally performed among patients with low BMD (n=41). A p-value <0.05 was considered statistically significant.

***Ethical considerations***

The study was conducted per institutional ethics committee approval, and patient confidentiality was maintained throughout.

## 3. Results

A total of 85 hemodialysis patients were enrolled. Key demographic and clinical characteristics are summarised in Table 1.

**Table 1:** Baseline demographic and clinical characteristics (n=85)

| Characteristic | Category | n | % |
|---|---|---|---|
| Age (years) | <50 | 21 | 24.7 |
| | 50–59 | 20 | 23.5 |
| | ≥60 | 44 | 51.8 |
| Gender | Male | 55 | 64.7 |
| | Female | 30 | 35.3 |
| Dialysis duration | <5 years | 70 | 82.4 |
| | ≥5 years | 15 | 17.6 |
| Comorbidity | HTN + T2DM | 38 | 44.7 |
| | HTN alone | 30 | 35.3 |
| | No comorbidity | 2 | 2.4 |
| Ca/phosphate binder use | No | 60 | 70.6 |
| | Yes | 25 | 29.4 |

Over half the cohort (51.8%) was aged ≥60 years, and males predominated (64.7%). Most patients (82.4%) had been on dialysis for less than 5 years. Combined hypertension and type 2 diabetes was the commonest comorbidity pattern (44.7%), and only 2.4% had no comorbidity. The majority (70.6%) were not on calcium supplements or phosphate binders.

***Bone mineral density and bone turnover***

Low BMD (osteopenia or osteoporosis) was found in 41/85 patients (48.2%): osteopenia in 34.1% and osteoporosis in 14.1% (Table 2, Figure 1). Bone turnover by iPTH was heterogeneous across the cohort, with no significant difference in turnover-category distribution between normal- and low-BMD groups (Table 3, Figure 2).

**Table 2:** Bone mineral density classification (n=85)

| BMD classification | n | % |
|---|---|---|
| Normal BMD | 44 | 51.8 |
| Osteopenia | 29 | 34.1 |
| Osteoporosis | 12 | 14.1 |

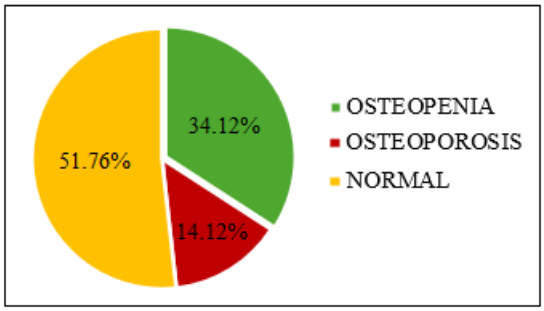


**Figure 1:** Distribution of bone mineral density among study participants (n=85)

**Table 3:** Bone turnover (iPTH) distribution by BMD status (n=85)

| Bone turnover (iPTH) | Total n(%) | Normal BMD n(%) | Low BMD n(%) |
|---|---|---|---|
| Low (<150 pg/mL) | 28 (33.0) | 13 (29.6) | 15 (36.6) |
| Normal (150–300 pg/mL) | 33 (38.8) | 19 (43.2) | 14 (34.2) |
| High (>300 pg/mL) | 24 (28.2) | 12 (27.2) | 12 (29.2) |

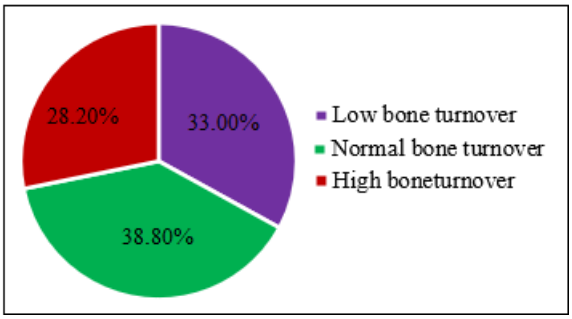


**Figure 2:** Distribution of bone turnover based on iPTH levels (n=85)

***Vitamin D status***

Vitamin D deficiency (<10 ng/mL) was present in 28.2% and insufficiency (10–29 ng/mL) in 51.8% of the cohort; only 20.0% had sufficient levels (≥30 ng/mL). Distribution was similar between normal- and low-BMD groups (Table 4, Figure 3), and the association between vitamin D and BMD status was not statistically significant ($\chi^2$=2.35, p=0.31).

**Table 4:** Distribution of serum vitamin D levels by BMD status (n=85)

| Vitamin D (ng/mL) | Total n (%) | Normal BMD n (%) | Low BMD n (%) |
|---|---|---|---|
| <10 | 24 (28.2) | 13 (29.6) | 11 (26.8) |
| 10–29 | 44 (51.8) | 25 (56.8) | 19 (46.4) |
| ≥30 | 17 (20.0) | 6 (13.6) | 11 (26.8) |

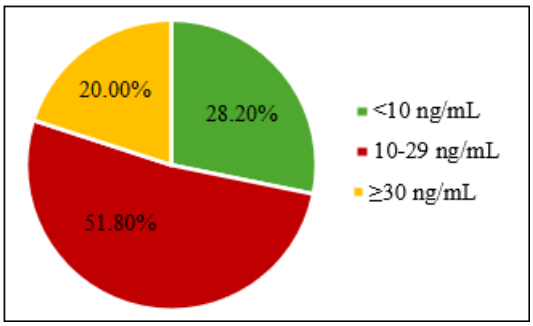


**Figure 3:** Distribution of participants by serum vitamin D levels (n=85)

***Serum phosphate- the key significant association***

Serum phosphate showed a statistically significant association with BMD status (Fisher's exact test, p=0.04) (Table 5). Interestingly, hyperphosphatemia (>4.5 mg/dL) was more frequent among patients with normal BMD (56.8%) than low BMD (34.2%), while normo-phosphatemia was more common in the low-BMD group (61.0%).

**Table 5:** Association between serum phosphate levels and BMD status (n=85)

| BMD status | Phosphate <2.5 mg/dL n(%) | Phosphate 2.5–4.5 mg/dL n(%) | Phosphate >4.5 mg/dL n(%) | P value |
|---|---|---|---|---|
| Normal BMD | 4 (9.1) | 15 (34.1) | 25 (56.8) | 0.04 |
| Low BMD | 2 (4.9) | 25 (61.0) | 14 (34.2) | |

No statistically significant association was found between bone turnover (iPTH category) and serum phosphate (p=0.85 overall; p=0.33 in the low-BMD subgroup) (Figure 4), serum calcium (p=0.23), ALP (p=0.42), dialysis duration (p=0.63), age (p=0.88), gender (p=0.55), or comorbidities (all p>0.05). Calcium/phosphate-binder use also showed no significant association with BMD or turnover status ($\chi^2$=0.25, p>0.05).

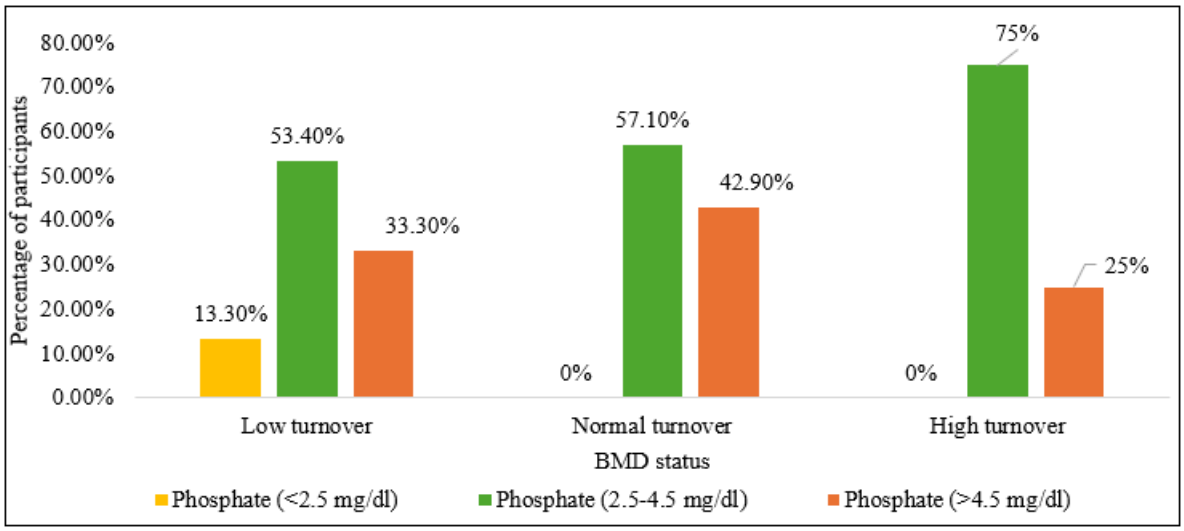


**Figure 4:** Association between serum phosphate levels and bone turnover (iPTH) among patients with low BMD (n=41)

**Table 5:** Association of co-morbidities with BMD Status (n=85)

| Co-morbidities | Normal BMD (T-score ≥ −1.0) N=44 | Low BMD (T-score < −1.0) N=41 | P value (Fischer exact t test) |
|---|---|---|---|
| No co-morbidity | 2 (4.6%) | 0 (0%) | 0.19 |
| HTN only | 13 (29.6%) | 17 (41.5%) | |
| T2DM only | 1 (2.2%) | 0 (0%) | |
| T2DM + HTN | 22 (50.0%) | 16 (39.0%) | |
| HTN + CAD | 0 (0%) | 3 (7.3%) | |
| T2DM + HTN + CAD | 3 (6.8%) | 3 (7.3%) | |
| T2DM + HTN + CVA | 1 (2.3%) | 2 (4.9%) | |
| T2DM + HTN + CAD + COPD | 2 (4.5%) | 0 (0%) | |

Patients with normal bone mineral density and low bone mineral density had similar distributions of co-morbidities. Of patients with normal bone mineral density (n = 44), T2DM with HTN was observed most frequently (n = 22, 50.0%) followed by HTN (n = 13, 29.6%). Of patients with low bone mineral density (n = 41), HTN was the most frequent co-morbidity (n = 17, 41.5%) followed by T2DM with HTN (n = 16, 39.0%). All other co-morbidity groups had low frequencies. There was no statistically significant difference between patient's co-morbidity distribution and bone mineral density groups (Fisher's exact test, p = 0.19).

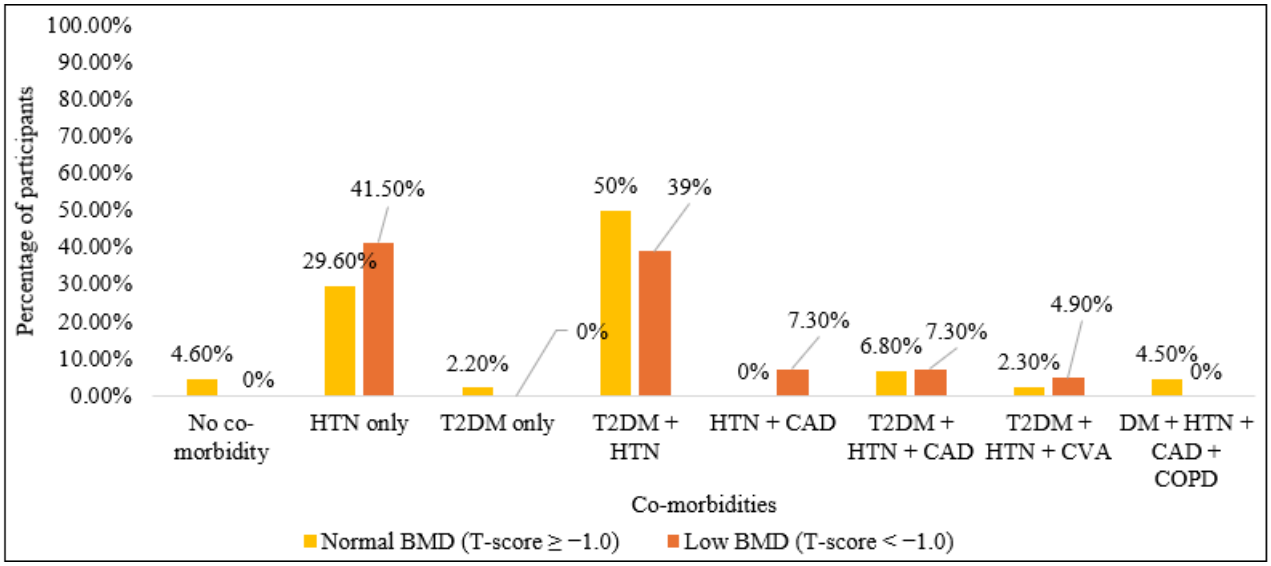


**Figure 5:** Association of co-morbidities with BMD Status (n=85)

**Table 6:** Association between serum Vitamin D levels and Bone Mineral Density (n=85)

| BMD levels | Vitamin D (<10 ng/ml) | Vitamin D (10-29 ng/ml) | Vitamin D (≥30 ng/ml) | Chi square | P value |
|---|---|---|---|---|---|
| Normal BMD (T-score ≥ −1.0) | 13 (29.6%) | 25 (56.8%) | 6 (13.6%) | 2.35 | 0.31 |
| Low BMD (T-score < −1.0) | 11 (26.8%) | 19 (46.4%) | 11 (26.8%) | | |

Among participants with normal BMD (T-score ≥ −1.0), 13 (29.6%) had severe vitamin D deficiency (<10 ng/mL), 25 (56.8%) had vitamin D levels between 10–29 ng/mL, and 6 (13.6%) had sufficient vitamin D levels (≥30 ng/mL). In contrast, among those with low BMD (T-score < −1.0), 11 participants (26.8%) had vitamin D levels <10 ng/mL, 19 (46.4%) had levels between 10–29 ng/mL, and 11 (26.8%) had sufficient vitamin D levels. The overall association between serum vitamin D levels and BMD status was not statistically significant ($\chi^2 = 2.35$, $p = 0.31$).

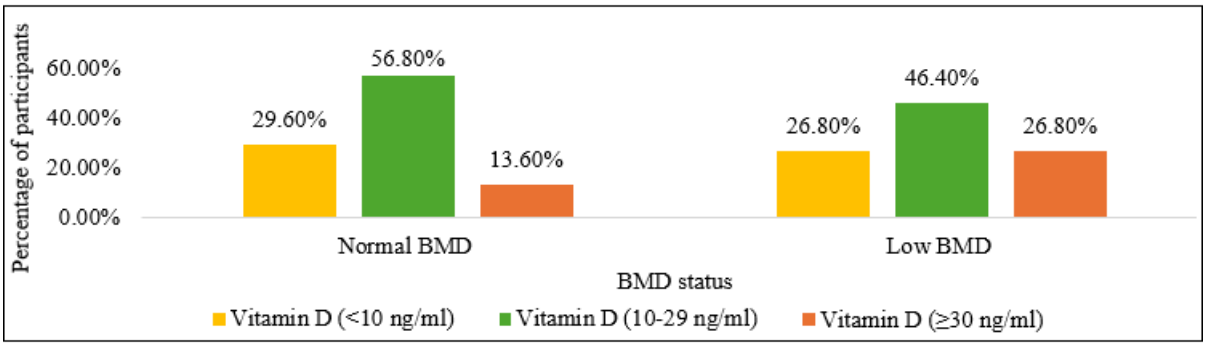


**Figure 6:** Association between serum Vitamin D levels and Bone Mineral Density (n=85)

**Table 7:** Association between Serum Phosphorus levels and Bone Mineral Density (n=85)

| BMD levels | Phosphate (<2.5 mg/dl) | Phosphate (2.5-4.5 mg/dl) | Phosphate (>4.5 mg/dl) | P value (Fischer exact t test) |
|---|---|---|---|---|
| Normal BMD (T-score ≥ −1.0) | 4 (9.1%) | 15 (34.1%) | 25 (56.8%) | 0.04 |
| Low BMD (T-score < −1.0) | 2 (4.9%) | 25 (61.0%) | 14 (34.2%) | |

Among participants with normal BMD (T-score ≥ −1.0), 4 (9.1%) had hypophosphatemia (<2.5 mg/dL), 15 (34.1%) had phosphate levels within the normal range (2.5–4.5 mg/dL), and the majority, 25 (56.8%), had hyperphosphatemia (>4.5 mg/dL). In contrast, among participants with low BMD (T-score < −1.0), 2 (4.9%) had hypophosphatemia, 25 (61.0%) had normal phosphate levels, and 14 (34.2%) had elevated phosphate levels. Overall, participants with normal BMD showed a higher proportion of hyperphosphatemia, whereas those with low BMD more commonly had phosphate levels within the normal range. This association between serum phosphate levels and BMD status was statistically significant (Fisher's exact test, $p = 0.04$).

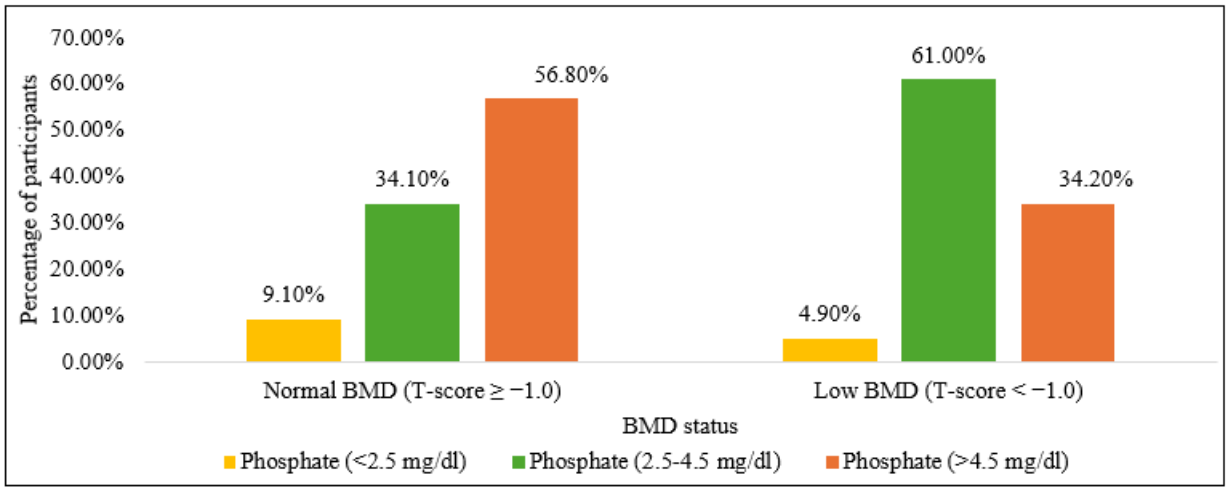


**Figure 7:** Association between Serum Phosphorus levels and Bone Mineral Density (n=85)

**Table 8:** Association between Serum Calcium and Bone Mineral Density (n=85)

| BMD levels | Calcium (<8.4 mg/dl) | Calcium (8.4-10.2 mg/dl) | Calcium (>10.2 mg/dl) | P value (Fischer exact t test) |
|---|---|---|---|---|
| Normal BMD (T-score ≥ −1.0) | 8 (18.2%) | 34 (77.3%) | 2 (4.5%) | 0.33 |
| Low BMD (T-score < −1.0) | 13 (31.7%) | 27 (65.9%) | 1 (2.4%) | |

Among participants with normal BMD (T-score ≥ −1.0), 8 (18.2%) had hypocalcaemia (<8.4 mg/dL), the majority 34 (77.3%) had calcium levels within the normal range (8.4–10.2 mg/dL), and 2 (4.5%) had hypercalcaemia (>10.2 mg/dL). In contrast, among participants with low BMD (T-score < −1.0), 13 (31.7%) had hypocalcaemia, 27 (65.9%) were normocalcaemic, and 1 (2.4%) had hypercalcaemia. Although hypocalcaemia was more frequently observed in participants with low BMD, the overall association between serum calcium levels and BMD status was not statistically significant (Fisher's exact test, p = 0.33).

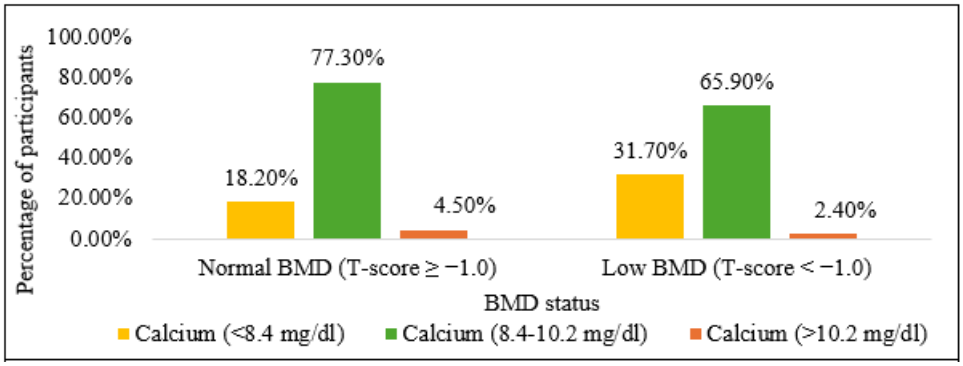


**Figure 8:** Association between Serum Calcium and Bone Mineral Density (n=85)

**Table 9:** Association between Use of Calcium Supplementation and Phosphate binders with BMD Status (n=85)

| BMD Status | Calcium Supplementation and Phosphate binders – Yes | Calcium Supplementation and Phosphate binders – No | Total | Chi square | P value |
|---|---|---|---|---|---|
| Normal BMD (T-score ≥ −1.0) | 14 (31.8%) | 30 (68.2%) | 44 (100%) | 0.25 | 0.61 |
| Low BMD (T-score < −1.0) | 11 (26.8%) | 30 (73.2%) | 41 (100%) | | |

Of the 44 patients with normal BMD, 14 (31.8%) were receiving calcium supplementation and phosphate binders, whereas 30 (68.2%) were not. Among the 41 patients with low BMD, 11 (26.8%) were on calcium supplementation and phosphate binders and 30 (73.2%) were not. Overall, calcium supplementation and phosphate binders was used in 29.4% of the study population.

There was no statistically significant association between calcium supplementation and phosphate binders and BMD status ($\chi^2 = 0.25$, df = 1, p = 0.61). Calcium supplementation and phosphate binders was used in 31.8% of patients with normal BMD and 26.8% of patients with low BMD.

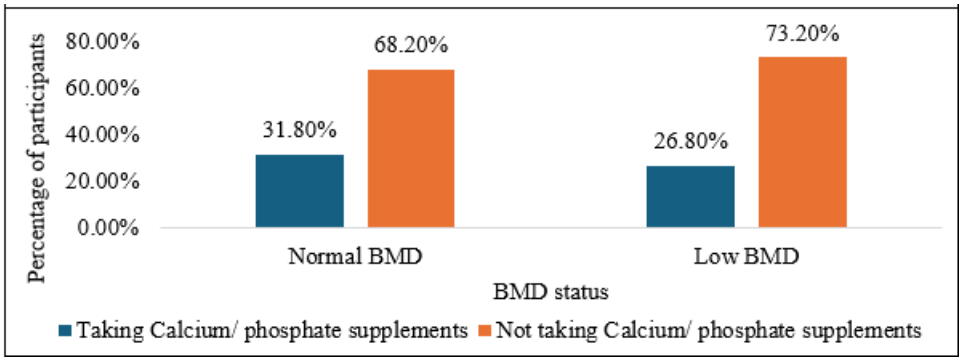


**Figure 9:** Association of Calcium Supplementation with BMD Status (n=85)

**Table 10:** Association between Duration of Dialysis and BMD status (n=85)

| BMD Status | <5 years | ≥5 years | Chi square | P value |
|---|---|---|---|---|
| Normal BMD (T-score ≥ −1.0) | 36 (81.8%) | 8 (18.2%) | 0.02 | 0.89 |
| Low BMD (T-score < −1.0) | 34 (82.9%) | 7 (17.1%) | | |

In the present study, among patients with normal bone mineral density (BMD) defined as a T-score ≥ −1.0, 36 patients (81.8%) had a dialysis duration of less than 5 years, while 8 patients (18.2%) had been on dialysis for 5 years or more. Among patients with low BMD, 34 (82.9%) had a dialysis duration of less than 5 years, while 7 patients (17.1%) had been on dialysis for 5 years or more. Statistical analysis revealed no significant association between duration of dialysis and normal BMD status (Chi-square = 0.02, p = 0.89).

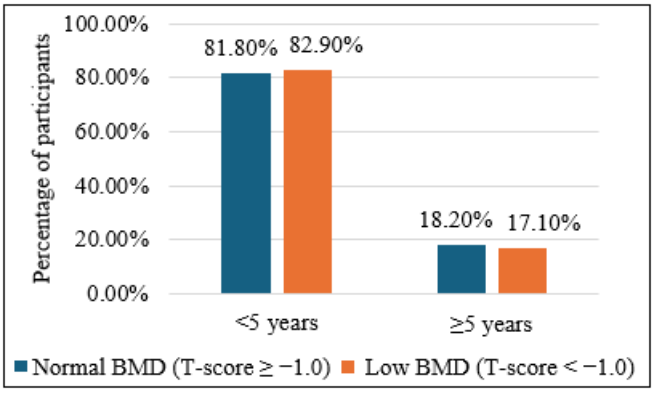


**Figure 10:** Association between Duration of Dialysis and BMD status (n=85)

## 4. Discussion

This prospective study found that nearly half (48.2%) of hemodialysis patients had reduced bone mineral density, with osteopenia more common than osteoporosis — suggesting early, progressive bone loss rather than advanced skeletal deterioration. This is broadly consistent with prior reports of high CKD-MBD burden in dialysis populations, though estimates vary by region and diagnostic modality.[4]

***Bone turnover heterogeneity***

Bone turnover by iPTH was distributed fairly evenly across low, normal, and high categories, consistent with the concept that CKD-MBD represents a spectrum rather than a single pathological entity.[6] Malluche et al. reported low-turnover disease in about half of dialysis patients on bone biopsy;[6] our iPTH-based estimate of low turnover (33.0%) is lower, which may reflect shorter dialysis vintage in our cohort (82.4% <5 years) and the known imperfect correlation between iPTH and bone histology.[10]

***Vitamin D- prevalent deficiency without a clear BMD signal***

Vitamin D deficiency/insufficiency was highly prevalent (80% of the cohort had levels <30 ng/mL), in keeping with earlier reports of 76–79% deficiency in CKD populations.[11] However, similar to Jamal et al.,[13] we found no significant association between vitamin D and BMD status (p=0.31), suggesting that PTH and phosphate dysregulation may play a more dominant role in skeletal remodelling than circulating 25-hydroxyvitamin D alone.

***Serum phosphate- the dominant biochemical correlate***

The significant association between serum phosphate and BMD status (p=0.04) is the key positive finding of this study. Notably, hyperphosphatemia was more frequent in the normal-BMD group, a seemingly paradoxical pattern that may reflect phosphate-driven stimulation of PTH and transient preservation of bone mineral density in earlier disease, before progressive skeletal deterioration sets in. Hyperphosphatemia has previously been associated to higher mortality risk independent of BMD;[15,16] our findings reinforce that phosphate control remains a central therapeutic target regardless of its direction of association with BMD.

***Comorbidities were not independent predictors***

Hypertension and diabetes were the dominant comorbidities in our cohort but showed no significant association with BMD status, echoing USRDS and DOPPS data suggesting that skeletal outcomes in CKD are driven more by biochemical and treatment-related factors than by comorbidity burden alone.[7,8]

## 5. Clinical implications

- Routine DEXA and biochemical screening (phosphate, PTH, vitamin D) should be considered in all maintenance hemodialysis patients, even early in the course of dialysis.
- Phosphate control deserves particular attention, given its significant and clinically actionable association with BMD status.
- Vitamin D and calcium supplementation alone are lessl likely to be sufficient to prevent bone loss and should be part of a broader metabolic monitoring strategy.

## 6. Limitations

- Single-centre design with modest sample size limits generalisability.
- Cross-sectional biochemical and BMD assessment precludes causal inference regarding bone turnover trajectories.
- Bone histology (gold standard) was not performed; iPTH-based turnover categorisation has known limitations.[10]

## 7. Conclusion

Reduced bone mineral density affects nearly half of hemodialysis patients, with osteopenia predominating over osteoporosis. Serum phosphate, not vitamin D or calcium, showed a statistically significant association with BMD status, underscoring disordered phosphate metabolism as a central, modifiable target in CKD-MBD. Effective management should combine periodic BMD assessment with strict phosphate control and comprehensive metabolic monitoring, rather than relying on calcium/vitamin D supplementation alone.

**Declarations**

***Ethics approval and consent to participate***
This study was approved by the Institutional Ethics Committee, Chinmaya Mission Hospital, Bengaluru . Written informed consent was obtained from all participants prior to enrolment, in accordance with the 2013 revision of the Declaration of Helsinki.

***Availability of data and materials***
The datasets generated and/or analysed during the current study are available from the corresponding author on reasonable request.

***Competing interests***
We declare that they have no competing interests, financial or non-financial, related to this work.

***Funding***
This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

***Authors' contributions***
Dr. Nandu Ernest: Conceptualization, Methodology, Data curation, Formal analysis, Investigation, Writing – original draft, Writing – review & editing.
Dr. Sridhara K M: Supervision, Conceptualization, Methodology, Validation, Writing – review & editing.

***Acknowledgements***
We thank the Department of General Medicine, Chinmaya Mission Hospital, and the patients who participated in this study.

## References

[1] Levin A, Tonelli M, Bonventre J, Coresh J, Donner JA, Fogo AB, et al; ISN Global Kidney Health Summit participants. Global kidney health 2017 and beyond: a roadmap for closing gaps in care, research, and policy. Lancet. 2017;390(10105):1888-1917.
[2] KDIGO CKD-MBD Update Work Group. KDIGO 2017 Clinical Practice Guideline Update for the Diagnosis, Evaluation, Prevention, and Treatment of Chronic Kidney Disease–Mineral and Bone Disorder (CKD-MBD). Kidney Int Suppl. 2017;7(1):1-59.
[3] Hou YC, Lu CL, Lu KC. Mineral bone disorders in chronic kidney disease. Nephrology (Carlton). 2018;23 Suppl 4:88-94.
[4] Malluche HH, Mawad H, Monier-Faugere MC. Renal osteodystrophy in the era of KDIGO guidelines: bone biopsy results from 630 dialysis patients. Clin J Am Soc Nephrol. 2011;6(4):905-914.
[5] Nasim A, Rafique Z, Talal A, Afzal A, Asrar A. Prevalence of Bone Mineral Disorder in Hemodialysis patients: A Single Centered Study of Local Population. Pak J Med Health Sci. 2022;16(07):134.
[6] Fukagawa M, Kido R, Komaba H, Onishi Y, Yamaguchi T, Tanaka M, et al. Abnormal mineral metabolism and mortality in hemodialysis patients with secondary hyperparathyroidism: Evidence from the MBD-5D study. Clin J Am Soc Nephrol. 2017;12(12):1938-1946.
[7] United States Renal Data System. USRDS 2022 Annual Data Report: Epidemiology of kidney disease in the United States. Am J Kidney Dis. 2023;81(3 Suppl 1):S1-S127.
[8] Young EW, Goodkin DA, Mapes DL, Port FK, Keen ML, Chen K, et al. The Dialysis Outcomes and Practice Patterns Study (DOPPS): an international hemodialysis study. Kidney Int. 2000;57(Suppl 74):S74-S81.
[9] Jha V, Garcia-Garcia G, Iseki K, Li Z, Naicker S, Plattner B, et al. Chronic kidney disease: global dimension and perspectives. Lancet. 2013;382(9888):260-272.
[10] Sprague SM, Bellorin-Font E, Jorgetti V, Carvalho AB, Malluche HH, Ferreira A, et al. Diagnostic accuracy of bone turnover markers and parathyroid hormone for predicting bone histology in patients with CKD treated by dialysis. Clin J Am Soc Nephrol. 2016;11(9):1553-1561.
[11] LaClair RE, Hellman RN, Karp SL, Kraus M, Ofner S, Li Q, et al. Prevalence of calcidiol deficiency in CKD stages 3–5. Am J Kidney Dis. 2005;46(5):877-883.
[12] Wolf M, Shah A, Gutierrez O, Ankers E, Monroy M, Tamez H, et al. Vitamin D levels and early mortality among incident hemodialysis patients. Kidney Int. 2007;72(8):1004-1013.
[13] Jamal SA, West SL, Nickolas TL, Miller PD. Fracture risk assessment in CKD stage 3–5. J Am Soc Nephrol. 2012;23(7):1146-1154.
[14] Kovesdy CP, Ahmadzadeh S, Anderson JE, Kalantar-Zadeh K. Association of activated vitamin D treatment and mortality in CKD. Arch Intern Med. 2008;168(4):397-403.
[15] Block GA, Hulbert-Shearon TE, Levin NW, Port FK. Association of serum phosphorus and calcium × phosphate product with mortality risk in chronic hemodialysis patients. Am J Kidney Dis. 2004;43(5):891-899.
[16] Tentori F, Blayney MJ, Albert JM, Gillespie BW, Kerr PG, Bommer J, et al. Mortality risk for dialysis patients

with different levels of serum calcium, phosphorus, and PTH: the Dialysis Outcomes and Practice Patterns Study (DOPPS). Am J Kidney Dis. 2008;52(3):519-530.

[17] Evenepoel P, Opdebeeck B, David K, D'Haese PC. Bone-Vascular Axis in Chronic Kidney Disease. Adv Chronic Kidney Dis. 2019;26(6):472-483.

[18] London GM. Bone-vascular axis in chronic kidney disease: a reality? Clin J Am Soc Nephrol. 2009;4(2):254-257.

[19] Moorthi RN, Moe SM. CKD-mineral and bone disorder: core curriculum 2011. Am J Kidney Dis. 2011;58(6):1022-1036.

[20] Shimada T, Hasegawa H, Yamazaki Y, Muto T, Hino R, Takeuchi Y, et al. FGF-23 is a potent regulator of vitamin D metabolism and phosphate homeostasis. J Bone Miner Res. 2004;19(3):429-435.